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EPIDEMIOLOGY BULLETIN

Robert B. Stroube, M.D., M.P.H., Health Commissioner Carl W. Armstrong, M.D., State Epidemiologist

Christopher Novak, M.D., M.P.H., Editor Vickie L. O'Dell, Layout Editor

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Varicella Zoster Virus Infection in Virginia

Introduction

Infection with varicella zoster virus (VZV) causes significant morbidity, and occasionally, mortality. Routine VZV immunization has been an important tool in reducing infection. However, it has also increased the number of atypical (but less severe) cases, and reduced physicians' overall experience in diagnosing varicella (chickenpox). There are also a number of barriers to immunization, including the belief that chickenpox is always a mild childhood illness, parental questions about safety and effectiveness of the vaccine, and concerns about waning immunity.²

In This Issue:

As a result, this article provides an update on VZV in Virginia to raise awareness of the continued presence of this pathogen and to support efforts to further reduce its impact. In particular, primary care providers should be aware of recent changes to vaccine recommendations, especially the use of two doses to decrease the risk of breakthrough infections.

VZV: Clinical Illness

Varicella

The varicella zoster virus is a member of the herpesvirus group. The virus is extremely infectious (although less contagious than measles, it is more contagious than mumps and rubella). Secondary attack rates among susceptible household contacts of persons with varicella can be as high as 90%.

VZV spreads to susceptible individuals through droplet, airborne, or direct contact with the respiratory secretions

of individuals with acute varicella VZV may also spread through contact with vesicular fluid from skin lesions of individuals with acute varicella or herpes zoster. The virus then enters through the respiratory tract and conjunctiva and replicates in the nasopharynx and the regional lymph nodes. *In utero* infection can occur by transplacental passage of virus during maternal varicella infection.³

A primary viremia follows 4–6 days after infection that disseminates the virus to other organs, such as the liver, spleen, and sensory ganglia. Further replication occurs in the viscera; a secondary viremia then leads to viral infection of the skin.¹

Overall, the incubation period for varicella is typically from 14 to 16 days after exposure (range: 10–21 days), but may be up to 28 days in immunocompromised patients and those who have received immune globulin therapy.¹

In children the rash is often the first sign of varicella; however, adults may experience 1–2 days of fever and malaise prior to rash onset. The period of communicability extends from 1 to 2 days **before** the onset of rash through the first 4 to 5 days after the rash appears, or until all lesions have formed crusts (VZV has not been isolated from crusted lesions). The rash

is generalized, pruritic, and classically progresses from macules to papules to vesicular lesions (superficial, delicate, 1–4 mm in diameter, containing a clear fluid on an erythematous base) that may rupture or become purulent before they dry and crust.1 The rash usually appears first on the scalp, then on the trunk, and finally on the extremities. Lesions also can occur on mucous membranes of the oropharynx, respiratory tract, vagina, conjunctiva, and the cornea.

Successive crops of lesions emerge over several days, and therefore appear in several stages of development. Healthy unvaccinated children usually have 200-500 lesions in 2 to 4 successive crops. Fever (up to 102°F), rash, and fatigue in children usually lasts 4 to 5 days. After primary infection the residual virus migrates to the sensorynerve ganglia to persist in the body as a latent infection.1

Breakthrough infection (i.e., varicella disease in a vaccinated person) is significantly milder, with fewer lesions (generally <50). Lesions are often maculopapular rather than vesicular.1 Most persons with breakthrough infection do not have fever, and the duration of illness is generally shorter.^{4,5} Mild breakthrough varicella cases are also significantly less contagious.⁵

Occasionally adults, and rarely children, develop complications from VZV infection that can include bacterial infection of the skin lesions, encephalitis, pneumonia, dehydration, or hepatitis.^{1,4} Underlying health conditions (e.g., malignancy, immune compromise) also predispose to severe complica-



tions. Rare complications of varicella include Reve syndrome, aseptic meningitis, transverse myelitis, Guillain-Barré syndrome, thrombocytopenia, hemorrhagic varicella, purpura fulminans, glomerulonephritis, myocarditis, arthritis, orchitis, uveitis, and iritis. Adults account for only 5% of reported cases of varicella but approximately 35% of deaths due to varicella.1

Fetal infection after maternal varicella during the first 20 weeks of gestation occasionally results in fetal death or embryopathy (congenital varicella syndrome).3 Infants born to mothers with onset of maternal varicella from 20 weeks gestation until approxi-

mately five days prior to delivery may have inapparent varicella, but usually have a benign course likely due to the passive transfer of maternal antibodies across the placenta. However, the onset of maternal

varicella from five days before to 48 hours after delivery may result in overwhelming infection of the neonate and a fatality rate as high as 30%. Such severe disease is believed to result from fetal exposure to VZV without the benefit of passive maternal antibody.1

Recovery from primary varicella infection usually results in lifetime immunity. In otherwise healthy persons a second occurrence of chickenpox is not common. However, as with other viral diseases, re-exposure to VZV may lead to re-infection that boosts antibody titers without causing clinical illness or detectable viremia.1

Public Health Reporting

Varicella is a notifiable condition in Virginia (however, herpes zoster is not).

Physicians, directors of laboratories, and directors of medical care facilities should contact their local health department to report any known or suspected cases of varicella, whether or not the individual has been previously vaccinated.

Herpes Zoster

Herpes zoster (shingles) is the result of recurrent VZV infection.4 Since declining cell-mediated immunity is the major determinant for VZV reactivation, herpes zoster typically occurs in those 50 years of age or older and in the immunocompromised.^{4,6} The condition is generally limited to a unilateral vesicular eruption in the distribution of a dermatome supplied by a dorsal root or extramedullary cranial nerve sensory ganglion. Most often, this involves the trunk or the area of the fifth cranial nerve.1

Two to four days prior to the eruption, there may be pain, itching, and paresthesia in the involved dermatome; there are few systemic symptoms, although patients may report headache, photophobia, and malaise. 1,7 An ery-

> thematous maculopapular rash then appears and progresses to clusters of clear vesicles that continue to form for three to five days and evolve through stages of pustulation, ulceration, and crusting. Healing occurs

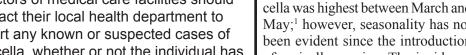
over a period of two to four weeks, and often results in scarring and permanent changes in skin pigmentation.⁷

Postherpetic neuralgia (PHN), or pain in the area of the recurrence after the lesions have resolved, can persist for months or years and lacks adequate therapy.1 The pain and discomfort can significantly diminish the patient's quality of life and ability to function.6 Ocular nerve and other organ involvement with herpes zoster can occur, often with severe sequelae. In immunocompromised persons, herpes zoster may disseminate, causing generalized skin lesions and central nervous system, pulmonary, and hepatic involvement.1

Epidemiology

United States

In the United States, in the prevaccine era, the incidence of varicella was highest between March and May; however, seasonality has not been evident since the introduction of varicella vaccine. The incidence



of herpes zoster does not follow a seasonal pattern.¹

In the pre-vaccine era, varicella was endemic in the United States, and virtually all persons acquired varicella by adulthood. Overall, there were approximately four million cases of chickenpox per year, with approximately 11,000 cases hospitalized. Hospitalization rates were approximately 2–3 per 1,000 cases among healthy children and

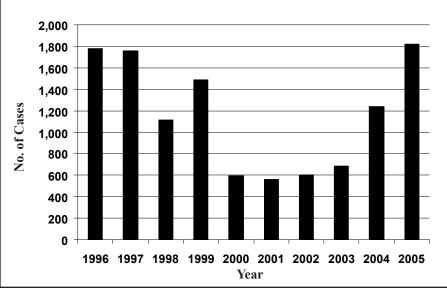
8 per 1,000 cases among adults. Death occurred in approximately 1 in 60,000 cases, although the risk of death was higher in adults (e.g., children 1–14 years of age: 1 death per 100,000 compared to adults 30–49 years of age: 25.2 deaths per 100,000 cases). From 1990 through 1996, an average of 103 deaths from varicella were reported each year. Most deaths occurred in immunologically normal children and adults.¹

Varicella vaccine became available in the U.S. in 1995. By 2004, cases of varicella had declined 83%–93%. Although the impact has been greatest among children 1–4 and 5–9 years of age, the decline has occurred in all age groups due to reduced exposure to the virus. Hospitalization rates declined 72% between 1995 and 2001 while mortality rates declined by approximately

75% in those 50 years of age or younger.8 Nationwide, varicella vaccination rates among those 12 to 35 months of age have continued to climb: in 2004, 87.5% of eligible children had been vaccinated.9

An estimated 300,000 episodes of herpes zoster occur annually. Ninety-five percent of these episodes are first occurrences, and 5% are recurrences. By 80 years of age, almost 15% of persons have experienced at least one

Figure 1. Reported Cases of Varicella in Virginia by Year (1996-2005)



episode of herpes zoster. However, the availability of a herpes zoster vaccine combined with reductions in naturally occurring VZV infections may reduce the impact of this condition over time.

Virginia

In 1999, Virginia schools began requiring a record of varicella vaccination prior to school entry for children born on or after January 1, 1997. As a result of immunization efforts, the incidence of varicella in the state has generally declined. From 1996 to 1999, an average of 1,536 cases of chickenpox were reported per year in Virginia. From 2000 to 2003 this average was 60% lower, at approximately 610 cases per year. However, in 2004 the number of reported cases increased significantly, and in 2005 there were 1,833 cases of varicella (including one death) reported in the

state of Virginia, a 149% increase over the previous five year average (Figure 1). It is thought that this represents increased reporting by both school nurses and physicians.

In 2005, the Northwest health region had a higher rate than the rest of the state (Figure 2), although the cause for this is not known.

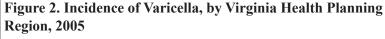
Of the reported cases in 2005, 784 (43%) were linked to one of 58 reported outbreaks, with some outbreaks as large as 55 identified cases

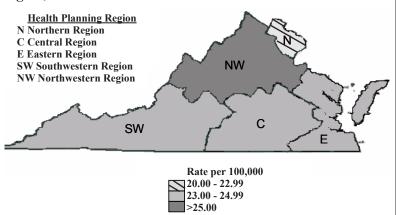
(mean = 13 cases).

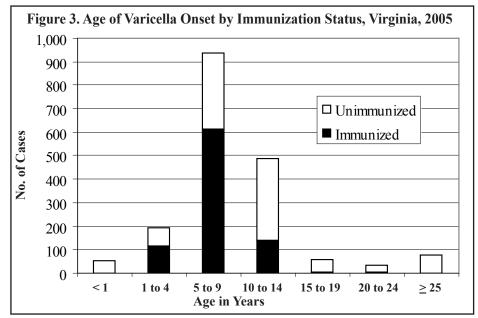
Overall, in Virginia in 2005, 935 (51%) reported cases occurred among children 5 to 9 years of age and 489 (27%) cases were among children 10 to 14 years of age. Of the 1,833 reported cases, 882 (48%) cases occurred in individuals with a history of age-appropriate vaccination: for children between 1 and 9 years of age, 731 (65%) cases had been appropriately immunized, while among cases 10 years of age or older, 151 (23%) occurred in appropriately immunized individuals (Figure 3). This reflects the short time period since the vaccine became available as well as the less common use of varicella vaccine in older age groups.

Of the reported cases of varicella in Virginia in 2005, illness duration data was available for 581 (32%) cases (Fig-

ure 4). The mean duration of illness for age-appropriate vaccinated cases was significantly lower (5.97 days versus 7.33 days) than the mean duration of illness for cases of inappropriately vaccinated, nonvaccinated, or unknown vaccination status (p<0.0001). Of the 1,540 cases for which data were reported, only 17 cases (1.1%) required hospitalization for varicella. While the numbers







are small, hospitalized cases appeared to be significantly older (mean = 16.9years) compared with non-hospitalized cases (mean = 9.4 years) (p<0.027) in 2005. In addition, immunization significantly reduced the risk of hospitalization in immunized persons, compared to inappropriately immunized, non-immunized, or individuals with unknown immunization status (RR = 0.067; 95% CI = 0.011-0.394). These findings are consistent with reports that varicella in older individuals is more likely to cause severe illness/complications, and that vaccination may protect against more severe illness.1

Polymerase chain reaction (PCR) is the method of choice for rapid clinical diagnosis. Direct fluorescent antibody (DFA) methods also exist, although they are less sensitive than PCR and require careful specimen collection and handling.¹

Specimens are best collected by unroofing a vesicle, preferably a fresh fluid-filled vesicle, and then rubbing the base of the lesion with a polyester swab. Crusts from lesions are also excellent specimens for PCR. A video developed for healthcare professionals to illustrate the most appropriate procedures for collecting skin lesion and blood specimens

for VZV testing can be viewed at www. cdc.gov/nip/diseases/varicella/surv/default.htm. Other specimen sources such as nasopharyngeal secretions, saliva, urine, bronchial washings, and cerebrospinal fluid are less desirable because positive test results from such specimens are much less likely.¹

Serologic testing of children is generally not necessary. However, serologic testing may be useful in adult vaccination programs. A variety of serologic tests for varicella antibody are available: enzyme-linked immunosorbent assay (EIA) is sensitive and specific, simple to perform, and widely available commercially. For the diagnosis of acute varicella infection, serologic confirmation would include a significant rise in varicella IgG. Testing with commercial kits for IgM antibody is not recommended since available methods lack sensitivity and false-positive IgM results are common in the presence of high IgG levels. Routine post-vaccination serologic testing is not recommended.1

Prevention

Prophylaxis of Varicella

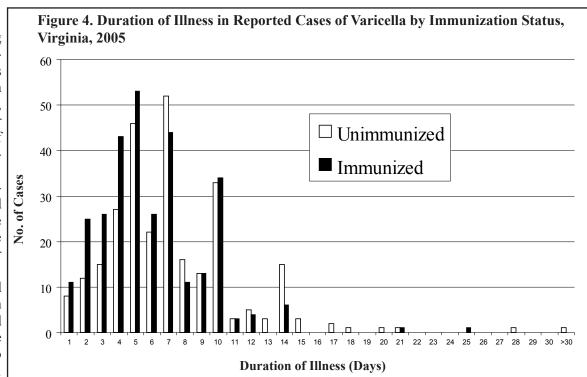
Varivax® (Merck) is a live attenuated vaccine developed and licensed for use in the United States in 1995. After one dose of single-antigen varicella vaccine, 97% of children 12 months to 12 years

Testing

Laboratory testing for varicella is not routinely required, but is useful for confirmation of the diagnosis (e.g., for atypical cases) or if determination of susceptibility is necessary.¹

Varicella zoster virus may be isolated in tissue culture. The most frequent source of isolation is vesicular fluid.¹

The use of rapid virus identification techniques is indicated for a case with severe or unusual disease to guide antiviral therapy.



of age develop detectable antibody titers. In field conditions, varicella vaccine is 80%–85% effective against infection and more than 95% effective against severe disease. Among healthy adolescents and adults, an average of 78% develop antibody after one dose, and 99% develop antibody after a second dose given 4 to 8 weeks later.¹

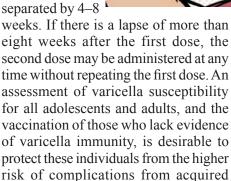
Currently, varicella vaccine is recommended for all children between 12 and 18 months of age who do not have contraindications. Although the vaccine may be given to all individuals within this age range regardless of prior history of varicella, vaccination is not necessary for children with a reliable history of varicella. A combined live attenuated measles-mumps-rubella and varicella (MMRV) vaccine (ProQuad®; Merck) for use in persons 12 months through 12 years of age is also available. Package inserts should be consulted for indications, contraindications, adverse events, vaccine storage and handling, and administration (available at: www. fda.gov/cber/label/varmer040505LBr. pdf).

In an effort to curb the recent trends in breakthrough varicella cases, the Advisory Committee on Immunization **Practices (ACIP) now recommends** a routine second dose of Varivax for children between 4 and 6 years of age. The second dose can be administered at an earlier age provided the interval between the first and second dose is at least three months. If the second dose is administered less than 28 days following the first dose, the second dose should be repeated.¹⁰ In addition, the ACIP has recommended that all children, adolescents, and adults who have previously received one dose of the vaccine should receive a second dose catch-up vaccination in order to ensure adequate immunity, particularly for adults who may experience more severe illness.^{2,10} The catch-up second dose can be administered at any interval longer than three months after the first dose.¹⁰

Varicella vaccine should also be administered to all adolescents and adults who do not have evidence of varicella immunity (e.g., documented age-appropriate immunization, history of varicella disease, laboratory evidence of

immunity, etc.). Persons 13 years of age and older without evidence of immunity should receive two doses of varicella vaccine separated by 4–8

varicella.1



The ACIP recommends that all healthcare workers should be immune to varicella, either from varicella disease or from vaccination. In healthcare settings, serologic screening of personnel who are uncertain of their varicella history, or who claim not to have had the disease, is likely to be cost-effective. 10

Women should be assessed prenatally for evidence of varicella immunity. Upon completion or termination of their pregnancies, women who do not have evidence of varicella immunity should receive the first dose of varicella vaccine before discharge from the healthcare facility. The second dose should be administered 4 to 8 weeks later (at the postpartum or other healthcare visit). To ensure administration of varicella vaccine, standing orders are recommended for healthcare settings where completion or termination of pregnancies occurs. 10

During a varicella outbreak, people who have received one dose of varicella vaccine should receive a second dose, provided the appropriate vaccination interval has elapsed since the first dose (three months for people 12 months to 12 years of age, and at least four weeks for people \geq 13 years of age).

VZV immunity following vaccination appears to be long-lasting, and is probably permanent in the majority of vaccinees. Some data also suggest that, among immunocompetent children, varicella vaccine reduces the risk of herpes zoster compared with those who have natural varicella infection.³

Prophylaxis of Herpes Zoster

One concern over varicella vaccination was that

the reduced periodic exposure of individuals to wild-type VZV virus could lead to waning of VZV-specific cell-mediated immunity, and therefore increase the incidence of herpes zoster. As a result, a more potent formulation of the varicella vaccine (Zostavax®; Merck) was developed to boost VZVspecific cellular immunity in individuals 55 years of age and older. Zostavax[®] has been found to reduce the overall incidence of herpes zoster by 51.3 percent and significantly reduced the pain and discomfort among subjects in whom herpes zoster developed.⁶ The vaccine is well tolerated, with the most common adverse event being a rash at the injection site. Package inserts should be consulted for indications, contraindications, adverse events, vaccine storage and handling, and administration (available at: www.fda.gov/cber/label/zosmer052506LB.htm).

Post-Exposure

Varicella vaccine is 70-100% effective in preventing illness or modifying the severity of illness if used within three days, and possibly up to five days, after exposure. Therefore, in the absence of contraindications to the vaccine, the ACIP recommends administering the varicella vaccine to individuals who do not have evidence of varicella immunity following an exposure to varicella. If exposure to varicella does not cause infection, postexposure vaccination should induce protection against subsequent exposure. If the exposure results in infection, there is no evidence that administration of varicella vaccine during the incubation period or prodromal stage of illness increases the risk for vaccine-associated adverse reactions. Varicella outbreaks in some settings (e.g., childcare facilities and schools) can persist for up to six months; varicella vaccine has been

used successfully to control outbreaks in these situations.¹

If a vaccinated healthcare worker is exposed to VZV, the employee should be monitored daily from day 10 to day 21 after exposure through the employee health program or infection control to determine clinical status: screen for fever, skin lesions, and systemic symptoms. As stated previously, persons with varicella may be infectious starting two days before rash onset. In addition, the healthcare worker should be instructed to immediately report fever, headache, or other constitutional symptoms and any skin lesions. The person should be placed on sick leave immediately if symptoms occur.1

For postexposure prophylaxis for varicella in contacts who do not have evidence of immunity and who are at high risk for severe disease or complications, varicella zoster immune globulin (VZIG) can be used. These contacts include:

- 1) immunocompromised individuals;
- neonates whose mothers develop signs and symptoms of varicella around the time of delivery (five days before to two days after);
- premature infants exposed during the neonatal period whose mothers do not have evidence of immunity;
- 4) premature infants who are less than 28 weeks gestation or who weigh less than 1,000 grams at birth and who are exposed during the neonatal period, regardless of maternal history of varicella; or,
- 5) pregnant women.¹

VZIG (VariZIGTM, Cangene Corporation) is available under an investigational new drug application (IND) submitted to the Food and Drug Administration (FDA). Treatment should be initiated as soon as possible after exposure; treatment after 96 hours is of uncertain value. In situations where administration of VariZIG does not appear possible within 96 hours of exposure, administration of immune globulin intravenous (IGIV) should be considered as an alternative. However, VZIG is preferred, since the titer of anti-varicella antibodies of any specific lot of IGIV is

variable. For pregnant women who cannot receive VariZIG within 96 hours of exposure, clinicians may choose either to administer IGIV or closely monitor the women for signs and symptoms of varicella and institute treatment with acyclovir if illness occurs.¹¹

Providers who identify a patient for whom VariZIG is indicated should contact FFF Enterprises (24-hour telephone, 800-843-7477). Under normal circumstances, VariZIG can be delivered from the distributor to its destination within 24 hours of request.¹¹

24 hours of request.

Antiviral drugs (e.g., acyclovir) are not recommended for routine post-exposure prophylaxis, but a seven day course beginning 7-10 days after exposure if vaccine is contraindicated may be used in some situations (e.g., susceptible immunocompromised patient exposed to varicella).³

Treatment

Patients with varicella should be under strict isolation precautions (standard, contact, and airborne) until all varicella lesions have crusted over. Immunized persons with breakthrough varicella with only maculopapular lesions should be isolated until no new lesions occur or the lesions have faded (complete resolution is not required).³ Newborns of mothers with rash onset five days before to two days after delivery should remain under strict quarantine for the entire incubation period (up to 28 days for those who receive VZIG or IGIV).^{1,3}

The most effective means of reducing the severity of VZV infection (varicella and herpes zoster) is administration of an antiviral drug.12 Several antiviral drugs are active against VZV, including acyclovir, valacyclovir, famciclovir, and foscarnet. Valacyclovir and famciclovir are approved for use only in adults. For varicella, clinical studies indicate that these drugs may be beneficial if given within 24 hours of onset of rash, reducing the number of days new lesions appear, the duration of fever, and the severity of cutaneous and systemic signs and symptoms. However, antiviral drugs have not been shown to decrease transmission of varicella, reduce the

duration of absence from school/work, or reduce complications.¹

The decision to use antiviral therapy, and the duration and route of therapy, should be determined by specific host factors, the extent of infection, and the initial response to therapy. The American Academy of Pediatrics does not recommend routine antiviral therapy for otherwise healthy infants or children with varicella. Oral acyclovir can be considered for otherwise healthy adolescents and adults, or secondary cases in a household, because of the increased risk of severe illness in these groups.³

Antiviral therapy may also be considered for persons with a chronic cutaneous or pulmonary disorder, persons receiving long-term salicylate therapy, and children receiving short, intermittent, or aerosolized courses of corticosteroids. If a child is immunocompromised, intravenous antiviral administration is indicated. Corticosteroids should be discontinued, if possible, after exposure. Oral acyclovir is not routinely recommended for pregnant women with uncomplicated varicella because the risks and benefits to the fetus and mother are not known. However, some experts recommend oral acyclovir for pregnant women with varicella, particularly during the second and third trimesters.1

For treatment of herpes zoster, individuals at highest risk for complications are elderly persons, those with herpes zoster ophthalmicus, and immunocompromised patients. Older age, a greater degree of skin-surface area involved, and more severe pain at presentation are all predictors of persistent pain. Patients meeting these criteria should be targeted for antiviral therapy. Adjunctive therapy with corticosteroids (e.g., prednisone) can be considered in older patients who have no contraindications.7 However, while antiviral therapy reduces the severity and duration of herpes zoster it does not prevent the development of postherpetic neuralgia.6

Drug therapy currently available for pain management in patients with PHN include topical agents (e.g., transdermal lidocaine, topical capsaicin), tricyclic antidepressants (TCAs; such as amitriptyline, desipramine, and nortriptyline) and possibly newer antidepressants (e.g., bupropion, venlafaxine), anticonvulsants (e.g., gabapentin, pregabalin), and opioid analgesics (e.g., oxycodone, methadone, morphine). Other therapies, such as nerve blocks or Transcutaneous Electrical Nerve Stimulation (TENS), may be considered. Psychological and behavioral interventions may also be introduced at any time in the course of treatment of PHN and are recommended as an integral part of treatment for patients with refractory PHN. 12

Conclusions

The number of reported varicella cases in Virginia increased in 2004 and 2005. This increasing trend is largely due to improved reporting by healthcare professionals and school personnel, as well as to changes in surveillance methods.

Reductions in the incidence of VZV infections are expected with the

implementation of a routine second dose. Healthcare professionals need to continue to promote the value of varicella vaccination. This is critical since provider recommendations are one of the primary determinants of patient vaccination, especially among adolescents and older patients.¹³ It is important for physicians to break down the barriers to immunization by avoiding missed opportunities, discussing immunization and disease history at office visits, and educating patients about the importance of varicella vaccination. Healthcare providers who have questions about vaccine preventable diseases may contact the Virginia Department of Health Division of Immunization at 804-864-8055.

Submitted by: Candace Hamm Virginia Commonwealth University

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Health Providers Surveyed on Their Practices Relating to Intimate Partner Violence



Intimate partner violence (IPV) is actual or threatened physical or sexual violence or psychological and emotional abuse directed toward a spouse, ex-spouse, current or former boyfriend or girlfriend, or current or former dating partner. Intimate partners may be heterosexual or of the same sex. Violence towards intimate partners is more prevalent among women in the United States than breast cancer, cervical cancer, or diabetes. Research suggests that healthcare professionals can play a critical role in mitigating the negative health consequences of IPV by using appropriate assessment and intervention procedures.

The Virginia Department of Health's Office of Family Health Services' Division of Injury and Violence Prevention has posted the results of its survey of Virginia

healthcare professionals' knowledge, attitudes, and behaviors concerning intimate partner violence. More than 2,000 healthcare professionals in a variety of settings responded to the survey.

Highlights of the report include:

- 64.8 percent of Virginia's health care providers reported not using IPV screening questions with any of their patients;
- 75.0 percent reported that, to their knowledge, their workplace did not have any written guidelines regarding IPV;
- 90.0 percent report never having been trained in IPV prevention; and
- Although 1 in 4 providers indicated that either they or someone close to them had been a victim of IPV, most estimated IPV prevalence in their practice to be "rare" or "very rare."

The full report of Virginia's 2006 Intimate Partner Violence Health Care Provider Survey contains detailed information about the survey, its results, specialty and practice-specific data, and recommendations for policy, procedures, and education. It is available at www.vahealth.org/civp/projectradarva/index.asp; to request a hard copy of the report, contact Laurie Crawford at 804-864-7705 or laurie.crawford@vdh.virginia.gov.



L	COMMUNICABL	E DISEASE REFERENCE	COMMUNICABLE DISEASE REFERENCE CHART FOR SCHOOL PERSONNEL	
=	NCUBATION PERIOD*	TRANSMISSION	COMMON SYMPTOMS	RECOMMENDATIONS
Fron	From 2-3 weeks, usually 14-16 days.	By direct contact with vesicular fluid or by airborne spread from respiratory tract secretions.	Sudden onset with slight fever and itchy eruptions which become vesicular (small blisters) within a few hours. Lesions commonly occur in successive crops, with several stages of maturity present at the same time. Communicable for as long as 5 days (usually 1-2 days) before eruption of vesicles and until all lesions are crusted (usually 5 days). Communicability may be prolonged in immunocompromised people.	CASE: Exclude from school for at least 5 days after eruptions first appear or until vesicles become dry. Avoid exposure to women in early pregnancy who have not had chickenpox and/or varicella vaccine. CONTACTS: On appearance of symptoms, exclude from school.
Usr	Usually 24-72 hours.	By contact with discharges from the conjunctivae or contaminated articles.	Pink or red eyeball with swelling of the eyelids and eye discharge. Eyelids may be matted shut after sleep. May involve one or both eyes.	CASE: Exclude from school while symptomatic or until 24 hours of antibiotic treatment has been completed. CONTACTS: School exclusion not indicated.
day day day Sa Sa Sa Sa USI	Campylobacteriosis: From 1-10 days, usually 2-5 days. E. coli O157:H7: From 2-10 days, usually 3-4 days. Glardiasis: From 3-25 days, usually 7-10 days. Salmonallosis: From 6-72 hours, usually 12-36 hours. Shigellosis: From 12-96 hours, usually 1-3 days.	By the fecal-oral route through direct contact or ingestion of contaminated food or water.	Ranges from sudden onset of fever, abdominal pain, diarrhea, nausea, and sometimes vomiting in salmonellosis, to cramps and bloody stools in severe cases of shigellosis and <i>E. coli</i> 0157: H7. Dangerous dehydration may occur in younger children. In giardiasis, persons may be asymptomatic or have decreased appetite and weight loss.	CASE: Exclude from school until cessation of acute diarrhea. Stress importance of proper handwashing. CONTACTS: School exclusion and stool cultures not indicated in absence of symptoms. Consult with your local health department for advice during suspected school outbreaks.
Fro	From 4-20 days.	Primarily through contact with respiratory secretions.	Rash characterized by a vivid reddening of the skin, especially of the face, which fades and recurs; classically, described as a "slapped face appearance." Mild symptoms of fever, body aches, and headache may occur 7-10 days before rash.	CASE: Exclusion from school not indicated. CONTACTS: School exclusion not indicated. Pregnant women and immunocompromised persons should seek medical advice.
L Frc	From 15-50 days, usually 28-30 days.	By the fecal-oral route through direct contact or ingestion of contaminated food or water.	Fever, loss of appetite, nausea, abdominal discomfort and weakness followed by jaundice. Many unrecognized mild cases without jaundice occur, especially in children. Communicability greatest from 7 days before to several days after onset of jaundice.	CASE: Exclude from school until physician advises return. Convalescence may be prolonged. CONTACTS: School exclusion not indicated. Stress importance of proper handwashing.
[년 Si	From 45-180 days, usually 60-90 days.	By direct contact with infected blood or body fluids. Transmission occurs when the hepatitis B virus enters the body through broken skin or mucous membranes.	Only a small proportion of acute infections have clinical symptoms. Symptoms are similar to those of hepatitis A.	CASE: Follow advice of child's physician and/or your local health department. CONTACTS: School exclusion not indicated.

DISEASE	INCUBATION PERIOD*	TRANSMISSION	COMMON SYMPTOMS	RECOMMENDATIONS
HIV Infection** and AIDS**	Variable	By direct contact with infected blood or body fluids. Transmission occurs when the human immunodeficiency virus enters the body through broken skin or mucous membranes.	A broad range of disease manifestations affecting multiple organ systems. Many children remain asymptomatic.	CASE: Follow advice of child's physician and/or your local health department. CONTACTS: School exclusion not indicated.
Measles** (Rubeola, Red Measles)	From 7-18 days, usually 14 days.	Airborne by droplet spread or direct contact with nasal or throat secretions of an infected person.	Prodrome characterized by fever followed by reddened eyes, runny nose, and cough. Dusky-red blotchy rash appears on day 3 or 4 and lasts 4 to 7 days. Highly communicable from one day before the beginning of symptoms to 4 days after the appearance of the rash.	CASE: Exclude from school until at least 4 days after appearance of the rash. Check immunization records of all students. Discuss with your local health department. CONTACTS: Exclude from school immediately on signs of prodrome.
Meningitis, bacterial (H. influenzae**, meningococcal**, pneumococcal)	H. influenzae: From 2-4 days Meningococcal: From 2-10 days, usually 3-4 days. Pneumococcal: From 1-4 days	By direct contact or droplet spread of nasopharyngeal secretions of an infected person.	Sudden onset of fever, headache, nausea, stiff neck and photophobia. Rash may occur in cases of meningococcal disease.	CASE: Exclude from school during acute illness. Non-communicable after 24-48 hours of appropriate drug therapy. CONTACTS: School exclusion not indicated. Discuss with your local health department to determine if close contacts need prophylactic treatment for <i>H. influenzae</i> and meningococcal forms.
Mumps**	From 14-25 days, usually 16-18 days.	By droplet spread or by direct contact with the saliva of an infected person.	Fever with swelling and tenderness of one or both parotid glands located below and in front of the ears. Unrecognized mild cases without swelling may occur. Communicable from 7 days before swelling until 9 days after.	CASE: Exclude from school for 9 days after the onset of parotid gland swelling. CONTACTS: School exclusion not indicated.
Pediculosis (Head Lice)	Under optimum conditions, eggs hatch in 7-10 days and reach maturity 1-3 weeks later.	By direct contact with an infested person or their personal belongings such as combs, brushes, and hats.	Severe itching and scratching, often with secondary infection. Eggs of head lice (nits) attach to hairs as small, round, gray lumps.	CASE: Exclude from school until treated. CONTACTS: Direct inspection of head. School exclusion not indicated in absence of infestation.
Pertussis**	From 6-20 days, usually 9-10 days.	By direct contact with respiratory secretions of an infected person by the airborne route.	The initial stage begins with upper respiratory symptoms and increasingly irritating cough. The paroxysmal stage usually follows within 1 to 2 weeks, and lasts 1 to 2 months. Paroxysmal stage is characterized by repeated episodes of violent cough broken by a high-pitched inspiratory whoop and vomiting. Older children may not have whoop. Convalescence may require many weeks.	CASE: Exclude from school until a physician advises return (usually 5 days after initiation of appropriate antibiotic therapy). Discuss with your local health department. CONTACTS: Exclude on first indication of symptoms.

DISEASE	INCUBATION PERIOD*	TRANSMISSION	COMMON SYMPTOMS	RECOMMENDATIONS
Ringworm of the Body (Tinea Corporis)	From 4 to 10 days.	By direct or indirect contact with lesions of an infected person or contaminated environmental surfaces.	Circular well-demarcated lesion that can involve face, trunk, or limbs. Itching is common.	CASE: Exclusion from school not indicated as long as lesions are covered or child is receiving treatment. During treatment, exclude from gymnasiums and swimming pools. CONTACTS: School exclusion not indicated.
Rubella** (German measles)	From 14 to 21 days, usually 14 to 17 days.	By direct contact or droplet spread of nasopharyngeal secretions of an infected person.	Mild symptoms; slight fever, rash of variable character lasting about 3 days; enlarged head and neck lymph glands common. Joint pain may occur, especially in older children and adults. Communicable for 7 days before onset of rash and at least 7 days thereafter.	CASE: Exclude from school for 7 days after onset of rash. Avoid exposure to women in early pregnancy. Check immunization records of all students. Discuss with your local health department. CONTACTS: Those who are pregnant and not immunized should be urged to seek medical advice.
Scabies	From 2 to 6 weeks.	By direct skin-to-skin contact.	Begins as itchy raised areas around finger webs, wrists, elbows, armpits, belt-line, and/or genitalia. Extensive scratching often results in secondary infection.	CASE: Exclude from school until 24 hours of antibiotic treatment has been completed. CONTACTS: Direct inspection of body. School exclusion not indicated in absence of infestation.
Streptococcal Diseases (Including Impetigo, Scarlet Fever, and "Strep" throat)	Variable, often 1-3 days, may be longer.	By direct contact with infected persons and carriers or by contact with their respiratory droplets.	Impetigo: Multiple skin lesions usually of exposed area (e.g., elbows, legs, and knees), but may involve any area. Lesions vary in size and shape, and begin as blisters, which rapidly mature into brown crusts on a reddened base. Healing from center outward produces circular areas, which may resemble ringworm. Scarlet Fever: Fever, sore throat, exudative tonsillitis or pharyngitis. Sandpaper-like rash appears most often on neck, chest, and skin folds of arms, elbows, groin, and inner aspect of thighs. "Strep" throat: Sudden onset of fever, sore throat, exudative tonsillitis or pharyngitis, and enlarged lymph nodes. Symptoms may be absent in some cases.	CASE: Exclude from school until lesions are healed or until 24 hours of antibiotic treatment has been completed. CONTACTS: Exclusion from school not indicated. Observe carefully for symptoms. CASE: Exclude from school during acute illness. Non-communicable after 24 hours of appropriate drug therapy. CONTACTS: Exclude on first indication of symptoms. Culturing of school contacts and treatment of carriers not usually indicated. CASE: Exclude from school until 24 hours of antibiotic treatment has been completed. CONTACTS: Exclusion from school not
	OF VINO VIDA GINGIFACIATI		NOTE: THE PRODUMENTALISMS APPLY ONLY TO SOLIDOL ACTOR OF BUILD FOR A SOCIED SHOWS CONSISTENCE OF	indicated. Observe carefully for symptoms.

NOTE: THESE RECOMMENDATIONS APPLY ONLY TO SCHOOL-AGED CHILDREN - A more complete discussion of these conditions and other communicable diseases may be found in Control of Communicable Diseases Manual (2004) published by the American Public Health Association and the Red Book 2003 Report of the Committee on Infectious Diseases published by the American Academy of Pediatrics. Additional information and consultation are also available through your local health department.

^{*} Based on the Control of Communicable Diseases Manual, 18th Edition (2004)

^{**} Officially reportable in Virginia to the local health department. All outbreaks and unusual occurrences of disease are also reportable.

Guidance for Individuals in Managing Dead Birds

Any sick or dead bird might be infected with one of a number of zoonotic diseases currently present in the United States, such as West Nile virus (WNV). If highly pathogenic H5N1 avian influenza A (H5N1) enters the US, it is likely to result in the death of wild birds. While no human infections with WNV have been linked to contact with live or dead wild birds in outdoor settings, and most human H5N1 cases overseas have been associated with close contact with infected poultry or their environment, healthcare professionals in Virginia may receive questions from their patients regarding exposures to dead birds. The following interim guidance has been developed by the Centers for Disease Control and Prevention (CDC) to guide the handling and management of dead birds

General Precautions for Collection of Single Dead Birds

When collecting dead birds, the risk of infection from WNV, H5N1, or any other pathogen may be eliminated by avoiding contamination of mucous membranes, eyes, and skin by material from the birds using the following safety precautions:

- When picking up any dead bird,
 - wear disposable impermeable gloves and place the bird directly into a plastic bag. Gloves should be changed if torn or otherwise damaged. If gloves are not available, use an inverted
 - double—plastic bag technique for picking up the carcass or scoop the carcass into a plastic bag using a shovel.
- In situations in which the bird carcass is in a wet environment

- or in other situations in which splashing or aerosolization of viral particles is likely to occur during disposal, safety goggles or glasses and a surgical mask may be worn to protect mucous membranes against splashed droplets or particles.
- Bird carcasses should be double bagged and placed in a trash receptacle that is secured from access by children and animals. If the carcass will be submitted for testing, hold it in a cool location until it can be delivered to authorities. Carcasses should not be held in close contact with food (e.g., not in a household refrigerator or picnic cooler).
- After handling any dead bird, the person should avoid touching his/ her face with gloved or unwashed hands.
- Any personal protective equipment (PPE) that was used (e.g., gloves, safety glasses, mask) should be discarded or disinfected* when done, and hands should then be washed with soap and water (or use an alcohol-based hand gel when soap and water are not available).
- If possible, before disposing of the bird, consult with a local animal control, health, wildlife, or agri-

cultural agency to inquire whether dead bird reports are being tallied and if the dead bird in question might be a candidate for testing. For local health department contact information

go to www.vdh.virginia.gov.

When collecting dead birds in higher-risk settings (e.g., when collecting large numbers or in confined indoor spaces, particularly once H5N1 has been confirmed in an area):



- Minimize any activities that generate airborne particles. For example, during the cleanup phase of the bird removal, avoid washing surfaces with pressurized water or cleaner (i.e., pressure washing), which could theoretically aerosolize viral particles that could then be inhaled. If aerosolization is unavoidable, the use of a filtering face-piece respirator (e.g., N-95) would be prudent, particularly while handling large quantities of dead birds repeatedly as part of regular work requirements.
- If using safety glasses, a mask, or a respirator, do not remove until after gloves have been removed and hands have been washed with soap and water (or use an alcohol-based hand gel when soap and water are not available). After PPE has been removed, hands should immediately be cleaned again. Personal protective equipment worn (e.g., gloves, mask, or clothing) should be disinfected* or discarded.

<u>Laboratory Biosafety</u> Recommendations

Laboratory handling of routine diagnostic specimens of avian carcasses requires a minimum of BSL-2 laboratory safety precautions. However, if a condition such as WNV or H5N1 infection is suspected, at a minimum BSL-3 precautions are advisable. Consult your institutional biosafety officer for specific recommendations.

Conclusions

The risk of infection from contact with sick or dead birds can be difficult to quantify and varies with the agent

(continued on Page 12)

For machine-washable, reusable PPE: Disinfect PPE in a washing machine with detergent in a normal wash cycle. Adding bleach will increase the speed of viral inactivation as will hot water but detergent alone in cold water will be effective. Follow manufacturer recommendations for drying the PPE. Non machine-washable, reusable PPE should be cleaned following the manufacturer's recommendations for cleaning.

^{*}Recommendations for PPE Disinfection

Total Cases Reported, August 2006

		Dogions					Total Cases Reported Statewide,		
				Regions			Jai	nuary - Augus	st
Disease	State	NW	N	SW	C	E	This Year	Last Year	5 Yr Avg
AIDS	71	2	37	4	15	13	369	370	454
Campylobacteriosis	59	12	16	17	6	8	393	370	418
Chickenpox	89	7	47	16	3	16	1,203	296	394
E. coli, Shiga toxin-producing	23	6	7	4	0	6	94	55	36
Giardiasis	27	5	12	1	5	4	261	354	262
Gonorrhea	681	39	56	105	162	319	4,304	5,593	6,279
Group A Strep, Invasive	10	3	3	1	0	3	98	67	67
Hepatitis, Viral									
Α	4	1	2	0	1	0	34	53	67
B, acute	9	0	3	3	2	1	36	102	122
C, acute	2	0	0	1	1	0	6	9	6
HIV Infection	96	5	23	6	36	26	605	486	573
Lead in Children [†]	117	18	12	30	39	18	431	330	445
Legionellosis	6	2	1	2	0	1	40	31	32
Lyme Disease	74	9	55	5	2	3	160	139	89
Measles	0	0	0	0	0	0	0	0	0
Meningococcal Infection	1	0	0	0	0	1	15	21	22
Pertussis	7	1	1	0	3	2	133	255	115
Rabies in Animals	43	10	9	11	5	8	405	341	335
Rocky Mountain Spotted Fever	29	2	4	13	5	5	69	48	23
Rubella	0	0	0	0	0	0	0	0	0
Salmonellosis	131	27	35	18	30	21	591	711	726
Shigellosis	7	1	5	0	1	0	42	85	255
Syphilis, Early [§]	43	5	14	2	4	18	215	174	144
Tuberculosis	23	4	11	0	7	1	174	197	176

Localities Reporting Animal Rabies This Month: Accomack 1 fox; Amelia 1 raccoon; Arlington 1 bat; Augusta 1 bat, 1 cat, 1 fox, 1 skunk; Bedford 1 raccoon; Bland 1 fox, 1 raccoon, 1 skunk; Carroll 1 fox, 1 raccoon; Chesapeake 1 raccoon; Fairfax 3 raccoons, 1 skunk; Fauquier 1 raccoon, 1 skunk; Hanover 1 skunk; Henrico 1 fox; Isle of Wight 1 fox; James City 1 raccoon; Loudoun 1 cat, 2 raccoons; Prince Edward 1 raccoon; Prince William 1 raccoon; Rappahannock 1 skunk; Richmond City 1 raccoon; Roanoke 1 skunk; Shenandoah 1 cat; Smyth 1 raccoon; Spotsylvania 1 skunk; Suffolk 1 raccoon; Virginia Beach 1 fox; Warren 1 raccoon; Wythe 1 groundhog, 2 skunks; York 2 raccoons.

Toxic Substance-related Illnesses: Adult Lead Exposure 7; Asbestosis 2; Pneumoconiosis 6.

(continued from Page 11)

and the situation. In general, proper handling of infected birds is unlikely to result in illness. Local public health officials can be consulted to help in selecting the most appropriate PPE for the situation.

Persons who develop an illness after handling sick or dead birds should seek medical attention. Their healthcare provider should report the incident to public health agencies if clinical symptoms or laboratory test results indicate possible infection (e.g., WNV, H5N1).

Source: Adapted from CDC. Interim Guidance for States Conducting Avian Mortality Surveillance for West Nile Virus (WNV) and/or Highly Pathogenic H5N1 Avian Influenza Virus. Available at: www.cdc. gov/flu/avian/doh/aviansurveillance.htm (Accessed: 09/11/2006).

Communicable Disease Reference Chart

(continued from page 10)

The Communicable Disease Reference Chart produced by the Virginia Department of Health (pages 8-10) was developed to provide basic guidance to school personnel on childhood illnesses and disease control, including exclusion. It is also available in a poster format through your local health department.

^{*}Data for 2006 are provisional. †Elevated blood lead levels ≥10µg/dL. §Includes primary, secondary, and early latent.